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NEWS 2	AUG 15	CAOLD to be discontinued on December 31, 2008
NEWS 3	OCT 07	EPFULL enhanced with full implementation of EPC2000
NEWS 4	OCT 07	Multiple databases enhanced for more flexible patent number searching
NEWS 5	OCT 22	Current-awareness alert (SDI) setup and editing enhanced
NEWS 6	OCT 22	WPIDS, WPINDEX, and WPIX enhanced with Canadian PCT Applications
NEWS 7	OCT 24	CHEMLIST enhanced with intermediate list of pre-registered REACH substances
NEWS 8	NOV 21	CAS patent coverage to include exemplified prophetic substances identified in English-, French-, German-, and Japanese-language basic patents from 2004-present
NEWS 9	NOV 26	MARPAT enhanced with FSORT command
NEWS 10	NOV 26	MEDLINE year-end processing temporarily halts availability of new fully-indexed citations
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NEWS EXPRESS JUNE 27 08 CURRENT WINDOWS VERSION IS V8.3,
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=> s ((kallikrein 8) or kallikrein8)
L1         152 ((KALLIKREIN 8) OR KALLIKREIN8)

=>
<------User Break------>

=> S (neuropsin or ovasin or TADG14)
L2         438 (NEUROPSIN OR OVASIN OR TADG14)

=> S L1 OR L2
L3         545 L1 OR L2

=> s l2 (10A) (Disease or disorder or condition or syndrome)
L4           9 L2 (10A) (DISEASE OR DISORDER OR CONDITION OR SYNDROME)

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L5           9 DUPLICATE REMOVE L4 (0 DUPLICATES REMOVED)

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reproductive)
L6           0 L2 (10A) (CARDIOVASCULAR OR NEUROLOGICAL OR METABOLIC OR UROLOG
               ICAL OR REPRODUCTIVE)

=> s l3 (10A) (Disease or disorder or condition or syndrome)
L7          10 L3 (10A) (DISEASE OR DISORDER OR CONDITION OR SYNDROME)

=> s l3 (10A) (cardiovascular or neurological or metabolic or urological or
reproductive)
L8           1 L3 (10A) (CARDIOVASCULAR OR NEUROLOGICAL OR METABOLIC OR UROLOG
               ICAL OR REPRODUCTIVE)

=> s l7 or l8
L9          11 L7 OR L8

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ENTER L# LIST OR (END):l8
PROCESSING COMPLETED FOR L8
L10          1 DUPLICATE REMOVE L8 (0 DUPLICATES REMOVED)
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ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):l7
PROCESSING COMPLETED FOR L7
L11          10 DUPLICATE REMOVE L7 (0 DUPLICATES REMOVED)

=> d l10 bib ab

L10  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2008 ACS on STN
AN   2005:216980  CAPLUS
DN   142:274082
TI   Diagnostics and therapeutics for diseases associated with human kallikrein
     8 (KLK8)
IN   Golz, Stefan; Brueggemeier, Ulf; Geerts, Andreas; Polej, Stefanie
PA   Bayer Healthcare AG, Germany
SO   PCT Int. Appl., 131 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     ---------------     ----   --------    --------------------    --------
PI   WO 2005022164        A2    20050310    WO 2004-EP9199          20040817
     WO 2005022164        A3    20050630
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
             EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
     EP 1664790           A2    20060607    EP 2004-764189          20040817
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK
     US 20070196372       A1    20070823    US 2006-568762          20060810
PRAI EP 2003-19799        A     20030830
     WO 2004-EP9199       W     20040817
AB   The invention provides a human kallikrein 8 (KLK8)
     which is associated with the cardiovascular diseases, dermatol.
     diseases, neurol. diseases, metabolic diseases, cancer disorders, urol.
     diseases, gastroenterol. diseases and reproduction disorders.  The invention
     also provides assays for the identification of compds. useful in the
     treatment or prevention of cardiovascular diseases, dermatol. diseases,
     neurol. diseases, metabolic diseases, cancer disorders, urol. diseases,
     gastroenterol. diseases and reproduction disorders.  The invention also
     features compds. which bind to and/or activate or inhibit the activity of
     KLK8 as well as pharmaceutical compns. comprising such compds.


=> d l11 1-10 bib ab

L11  ANSWER 1 OF 10  CAPLUS  COPYRIGHT 2008 ACS on STN
AN   2008:458215  CAPLUS
DN   149:50558
TI   Correlation between SPINK5 Gene Mutations and Clinical Manifestations in
     Netherton Syndrome Patients
AU   Komatsu, Nahoko; Saijoh, Kiyofumi; Jayakumar, Arumugam; Clayman, Gary L.;
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Tohyama, Mikiko; Suga, Yasushi; Mizuno, Yuki; Tsukamoto, Katsuhiko; Taniuchi, Katsushige; Takehara, Kazuhiko; Diamandis, Eleftherios P.
CS Department of Pathology and Laboratory Medicine, Mount Sinai Hospital, Toronto, ON, Can.
SO Journal of Investigative Dermatology (2008), 128(5), 1148-1159
CODEN: JIDEAE; ISSN: 0022-202X
PB Nature Publishing Group
DT Journal
LA English
AB Netherton syndrome (NS) is a congenital ichthyosiform dermatosis caused by serine protease inhibitor Kazal-type 5 (SPINK5) mutations. Tissue kallikreins (KLKs) and lymphoepithelial Kazal-type-related inhibitor (LEKTI) (SPINK5 product) may contribute to the balance of serine proteases/inhibitors in skin and influence skin barrier function and desquamation. SPINK5 mutations, causing NS, lead to truncated LEKTI; each NS patient possesses LEKTI of a different length, depending on the location of mutations. This study aims to elucidate genotype/phenotype correlations in Japanese NS patients and to characterize the functions of each LEKTI domain. Since the authors were unable to demonstrate truncated proteins in tissue from patients with NS, the authors used recombinant protein to test the hypothesis that the length of LEKTI correlated with protease inhibitory activity. Genotype/phenotype correlations were observed with cutaneous severity, growth retardation, skin infection, stratum corneum (SC) protease activities, and KLK levels in the SC. Predominant inhibition by LEKTI domains against overall SC protease activities was trypsin-like (Phe-Ser-Arg-) activity by LEKTI domains 6-12, plasmin- and trypsin-like (Pro-Phe-Arg-) activities by domains 12-15, chymotrypsin-like activity by all domains, and furin-like activity by none. KLK levels were significantly elevated in the SC and serum of NS patients. These data link LEKTI domain deficiency and clin. manifestations in NS patients and pinpoints to possibilities for targeted therapeutic interventions.
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:897857 CAPLUS
DN 147:403500
TI Neuropsin promotes oligodendrocyte death, demyelination and axonal degeneration after spinal cord injury
AU Terayama, R.; Bando, Y.; Murakami, K.; Kato, K.; Kishibe, M.; Yoshida, S.
CS Department of Functional Anatomy and Neuroscience, Asahikawa Medical College, Asahikawa, 078-8510, Japan
SO Neuroscience (New York, NY, United States) (2007), 148(1), 175-187
CODEN: NRSCDN; ISSN: 0306-4522
PB Elsevier
DT Journal
LA English
AB Previous studies indicated that the expression of neuropsin, a serine protease, is induced in mature oligodendrocytes after injury to the CNS. The pathophysiol. of spinal cord injury (SCI) involves primary and secondary mechanisms, the latter contributing further to permanent losses of function. To explore the role of neuropsin after SCI, histochem. and behavioral analyses were performed in wild-type (WT) and neuropsin-deficient (neuropsin-/-) mice using a crush injury model, a well-characterized and consistently reproducible model of SCI. In situ hybridization revealed that neuropsin mRNA expression was induced in the spinal cord white matter from WT mice after crush SCI, peaking at day 4. Neuropsin-/- mice showed attenuated demyelination, oligodendrocyte death, and axonal damage after SCI. Although axonal degeneration in the corticospinal tract was obvious caudal to the lesion site in both strains of mice after SCI, the number of surviving nerve fibers caudal to the lesion

was significantly larger in neuropsin-/- mice than WT mice. Behavioral anal. revealed that the recovery at days 10-42 was significantly improved in neuropsin-/- mice compared with WT mice in spite of the severe initial hindlimb impairments due to SCI in both strains. These observations suggest that neuropsin is involved in the secondary phase of the pathogenesis of SCI mediated by demyelination, oligodendrocyte death, and axonal degeneration.

RE.CNT 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:437557 CAPLUS
DN 144:466059
TI Genes showing changes in levels of expression in neurological diseases and their use in early diagnosis and in monitoring of treatment
IN Scherzer, Clemens R.; Gullans, Steven R.; Jensen, Roderick V.
PA Brigham and Women's Hospital, Inc., USA
SO PCT Int. Appl., 118 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2006050475	A2	20060511	WO 2005-US39876	20051103
	WO 2006050475	A3	20060908		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	US 20060134664	A1	20060622	US 2005-266774	20051103
PRAI	US 2004-624592P	P	20041103		
	US 2005-645423P	P	20050119		

AB Genes showing changes in levels of expression in neurodegenerative diseases (ND) are identified for use in diagnosis and in monitoring of treatments. In addition, these genes identify therapeutic targets, the modification of which may prevent ND development or progression. Identification genes associated with Parkinson's disease, Alzheimer's disease, and supranuclear palsy is reported.

L11 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:238155 CAPLUS
DN 144:310062
TI Genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer
IN Kloeppel, Guenter; Luettges, Jutta; Kalthoff, Holger; Ammerpohl, Ole; Gruetzmann, Robert; Pilarsky, Christian; Saeger, Hans Detlev; Alldinger, Ingo
PA Technische Universitaet Dresden, Germany
SO Ger. Offen., 132 pp.
CODEN: GWXXBX
DT Patent
LA German

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 102004042822	A1	20060316	DE 2004-102004042822	20040831
	WO 2006024283	A2	20060309	WO 2005-DE1527	20050826
	WO 2006024283	A3	20060831		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

	DE 112005002742	B4	20080521	DE 2005-112005002742	20050826
PRAI	DE 2004-102004042822	A	20040831		
	WO 2005-DE1527	W	20050826		

AB Genes showing altered levels of expression in healthy vs. neoplastic pancreas are identified for use in the diagnosis of cancers including ductal adenocarcinoma; as indicators in screening for effective drugs; and as targets for nucleic acid-based therapies including antisense nucleic acids or siRNA. Gene expression profiling identified 1419 genes showing changes in levels of expression in neoplastic epithelium of which 650 were up-regulated and 769 were down-regulated. Of the 1419 genes, 1267 were not previously known to have any connection with pancreatic neoplasms.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:205510 CAPLUS
DN 145:207574
TI Human Kallikrein 8 Protein Is a Favorable Prognostic Marker in Ovarian Cancer
AU Borgono, Carla A.; Kishi, Tadaaki; Scorilas, Andreas; Harbeck, Nadia; Dorn, Julia; Schmalfeldt, Barbara; Schmitt, Manfred; Diamandis, Eleftherios P.
CS Department of Pathology and Laboratory Medicine, Mount Sinai Hospital, University of Toronto, Toronto, ON, Can.
SO Clinical Cancer Research (2006), 12(5), 1487-1493
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal
LA English
AB Human kallikrein 8 (hK8/neuropsin/ovasin; encoded by KLK8) is a steroid hormone-regulated secreted serine protease differentially expressed in ovarian carcinoma. KLK8 mRNA levels are associated with a favorable patient prognosis and hK8 protein levels are elevated in the sera of 62% ovarian cancer patients, suggesting that KLK8/hK8 is a prospective biomarker. Given the above, the aim of the present study was to determine if tissue hK8 bears any prognostic significance in ovarian cancer. Using a newly developed ELISA, hK8 was quantified in 136 ovarian tumor exts. and correlated with clinicopathol. variables and outcome [progression-free survival (PFS); overall survival (OS)] over a median follow-up period of 42 mo. hK8 levels in ovarian tumor cytosols ranged from 0 to 478 ng/mg total protein, with a median of 30 ng/mg. An optimal cutoff value of 25.8 ng/mg total protein (74th percentile) was selected based on the ability of hK8 values to predict the PFS of the study population and to categorize

tumors as hK8 pos. or neg. Women with hK8-pos. tumors most often had lower-grade tumors (G1), no residual tumor after surgery, and optimal debulking success ($P < 0.05$). Univariate and multivariate analyses revealed that patients with hK8-pos. tumors had a significantly longer PFS and OS than hK8-neg. patients ($P < 0.05$). Kaplan-Meier survival curves further confirmed a reduced risk of relapse and death in women with hK8-pos. tumors ($P = 0.001$ and $P = 0.014$, resp.). These results indicate that hK8 is an independent marker of favorable prognosis in ovarian cancer.

RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:930483 CAPLUS
DN 145:486901
TI Disease processes may be reflected by correlations among tissue kallikrein proteases but not with proteolytic factors uPA and PAI-1 in primary ovarian carcinoma
AU Dorn, Julia; Harbeck, Nadia; Kates, Ronald; Magdolen, Viktor; Grass, Linda; Soosaipillai, Antoninus; Schmalfeldt, Barbara; Diamandis, Eleftherios P.; Schmitt, Manfred
CS Clinical Research Unit, Department of Obstetrics and Gynecology, Technical University of Munich, Munich, D-81675, Germany
SO Biological Chemistry (2006), 387(8), 1121-1128
CODEN: BICHF3; ISSN: 1431-6730
PB Walter de Gruyter GmbH & Co. KG
DT Journal
LA English
AB In epithelial ovarian cancer, the high mortality rate is usually ascribed to late diagnosis, since these tumors commonly lack early-warning symptoms, but tumor-associated biomarkers useful for prognosis or therapy response prediction are in short supply. However, members of the tissue kallikrein serine protease family, the serine protease uPA and its inhibitor PAI-1, are associated with tumor progression of ovarian cancer. Therefore, we used ELISA to determine uPA, PAI-1, and tissue kallikreins hK5-8, 10, 11, and 13 in exts. of 142 primary tumor tissue specimens from ovarian cancer patients and studied the strength of association between protein expression levels of these tumor tissue-associated factors. UPA, PAI-1, hk5, and hk8 were related to FIGO stage; hK5 expression was higher in FIGO III/IV than in FIGO I/II patient tissues. PAI-1 and hk5 differed significantly according to nuclear grading; expression of hK5 was higher in G3 than in G1/2 tumors. Assocns. between uPA, PAI-1, and the tissue kallikreins were weak. There were strong pairwise correlations within the cluster of tissue kallikreins hK5, 6, 7, 8, 10, and 11, but their bivariate distributions depended on nuclear grading. These results support the notion that several tissue kallikreins are co-expressed in ovarian cancer patients, substantiating the existence of a steroid hormone-driven tissue kallikrein cascade in this disease.
RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2002:784956 CAPLUS
DN 138:71092
TI Epidermal expression of serine protease, neuropsin (KLK8) in normal and pathological skin samples
AU Kuwae, K.; Matsumoto-Miyai, K.; Yoshida, S.; Sadayama, T.; Yoshikawa, K.; Hosokawa, K.; Shiosaka, S.
CS Division of Structural Cell Biology, Nara Institute of Science and Technology, Nara, 630-0101, Japan
SO Molecular Pathology (2002), 55(4), 235-241

CODEN: MOPAF6; ISSN: 1366-8714
PB BMJ Publishing Group
DT Journal
LA English
AB The expression of human neuropsin (KLK8) mRNA in normal and pathol. skin samples was analyzed and the results compared with those for tissue plasminogen activator (tPA) mRNA. Northern blot and in situ hybridization analyses of KLK8 mRNA in normal and lesional skin of patients with cutaneous diseases were performed. A weak signal for KLK8 mRNA and no signal for tPA mRNA was seen in normal skin on northern blot anal. Weak signals for KLK8 were localized to the superficial cells beneath the cornified layer in normal skin on in situ hybridization. Psoriasis vulgaris, seborrheic keratosis, lichen planus, and squamous cell carcinoma skin samples, which show severe hyperkeratosis, displayed a high d. of KLK8 mRNA on Northern and in situ hybridization analyses. The signals were localized in granular and spinous layers of lesional skin in all hyperkeratic samples, including the area surrounding the horn pearls of squamous cell carcinoma. To examine the relation between mRNA expression and terminal differentiation, the expression of KLK8 mRNA was analyzed in cell cultures. When keratinisation proceeded in high calcium medium, a correlative increase in the expression of KLK8 mRNA was observed The results are consistent with a role for this protease in the terminal differentiation of keratinocytes.
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2001:219937 CAPLUS
DN 135:316834
TI Expression of neuropsin in oligodendrocytes after injury to the CNS
AU He, X.-P.; Shiosaka, S.; Yoshida, S.
CS Division of Structural Cell Biology, 8916-5 Takayama, Nara Institute of Science and Technology, Nara, Ikoma, 630-0101, Japan
SO Neuroscience Research (Shannon, Ireland) (2001), 39(4), 455-462
CODEN: NERADN; ISSN: 0168-0102
PB Elsevier Science Ireland Ltd.
DT Journal
LA English
AB Proteases are involved in a variety of processes including demyelination after injury to the central nervous system. Neuropsin is a serine protease, which is constitutively expressed in the neurons of the limbic system. In the present study, intrahippocampal kainate injection and enucleation were performed on adult mice. Neuropsin mRNA and protein expression was detected by in situ hybridization and immunohistochem. Double in situ hybridization confirmed that the mRNA expression was induced in oligodendrocytes. One day after kainate injection to the hippocampus, neuropsin mRNA was expressed, peaking 4-8 days postoperatively and disappearing at 14 days. Immunohistochem. and immunoelectron microscopy revealed that neuropsin was expressed in the cell body of oligodendrocytes and myelin. To see if neuropsin degrades myelin protein, purified myelin was incubated with recombinant neuropsin. A decrease in the intensity of the bands of myelin basic protein was observed These results indicate that neuropsin is involved in demyelination.
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1999:744266 CAPLUS
DN 132:2410
TI Cloning of cDNA for and promoter of human neuropsin
IN Shiosaka, Sadao; Yoshida, Shigetaka

PA Igaku Seibutsugaku Kenkyusho K. K., Japan
SO Jpn. Kokai Tokkyo Koho, 12 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 11318461	A	19991124	JP 1998-133615	19980515
PRAI	JP 1998-133615		19980515		

AB The cDNA encoding a 260-amino-acid neuropsin that specifically expressed in hippocampus was isolated. The cDNA consists of 6 defined exons. Also provided are the promoter region and 8 oligonucleotide primers for the neuropsin. Neuropsin is useful for the studies of brain diseases and functions.

L11 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1996:738171 CAPLUS
DN 126:6455
OREF 126:1495a,1498a
TI Antibody against neuropsin
IN Shiosaka, Sadao
PA Igaku Seibutsugaku Kenkyusho K, Japan; Medical and Biological Laboratories Co., Ltd.
SO Jpn. Kokai Tokkyo Koho, 9 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 08245700	A	19960924	JP 1995-83154	19950314
	JP 3663228	B2	20050622		
PRAI	JP 1995-83154		19950314		

AB Disclosed is an antibodies against a novel brain hippocampus-specific neuropsin. The antibody is for immunoblotting anal. of neuropsin in brain tissue, for study of mechanism of memory and learning and memory loss (e.g. Alzheimer's disease), and for therapy of brain function insufficiency (e.g. epilepsy), etc. Thus, pVL1392 encoding the novel neuropsin was expressed in insect cell High 5, purified neuropsin was used to raise antibody in New Zealand white rabbit, and the antibody was used in immunoassay to identify neuropsin.

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